

Syllable Structure, Syllable Duration and Final Lengthening in Parkinsonian French Speech

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Key Words: Parkinson's disease, syllable duration, syllable structure, final lengthening

ABSTRACT

The present study investigates the duration of syllables with relation to position within phrases and the pattern of segment omissions within syllables in a text read by 12 French PD patients and 12 French control subjects. Three main tendencies emerged. The first was similar duration of syllables in PD and control speech, which may result from a combination of articulatory undershoot and slowness of speech gestures. The second was a normal incidence of segment omissions in both groups: these were mostly coda consonants and/or the second member of C1C2 sequences. The third was a normal production and a strong correlation of final lengthening with the syntactic structure of sentences in both PD speech and control speech. Having analysed the results the study evaluates their implication with respect to the role of basal ganglia in the production of speech.

INTRODUCTION

Speakers make temporal adjustments at each level of speech production to convey linguistic information. Hence disorders in motor control such as those associated with Parkinson's disease (PD), a progressive destruction of dopamine-producing neurons within the striatum of the basal ganglia, can be expected to affect the temporal organization of speech. This has been already confirmed by numerous perceptual and acoustic investigations all of which reported abnormalities of temporal speech patterns subsequent to PD. For example, in their perceptual ratings of the parkinsonian dysarthria, Darley, Aronson and Brown (1969) defined two major clusters of deviant dimensions : phonatory incompetence and prosodic insufficiency, which comprises of monotony of pitch and loudness, reduced stress, short phrases, variable rate, short rushes of speech. Later on, in a correlational study of vocal and clinical symptoms in 81 French PD patients, Segulier, Spira, Dordain and Chevrie-Muller (1974) reported that speech characteristics bearing the closest relationship to clinical symptoms were : (1) time factors involved in speaking rate and monotony, and (2) changes in voice quality. More recent studies on intelligibility in PD French speech (Viallet and Gentil, 2001; Ozsancak, Parais, and Auzou, 2002) confirmed the salient role of speech rate abnormalities in intelligibility loss; in addition Viallet and Gentil (2001) observed that intonation and speech rate are among the first speech components affected by PD.

While there is agreement on the impact of PD on speech rate, there is less unanimity on how PD affects speech rate and its components (pause time and articulation rate). In comparisons of readings of texts by PD patients and age-matched control subjects, some PD patients experienced speech acceleration (Canter, 1963; Darley, Aronson and Brown, 1969) while others were found to speak more slowly than normal (Gräber et al., 2002). Furthermore, studies of pause time reported shorter breath groups and longer, more frequent pauses in the speech of

PD subjects (Duez, 2005; Hammen and Yorkston, 1996; Metter and Hanson, 1986; Solomon and Hixon, 1993). However, other studies indicated no significant pause duration differences between PD and control speakers (Volkman, Hefter, Lange and Freund, 1992). At habitual speaking rate, articulation rate was found to be faster (Hammen and Yorkston, 1996) or about the same (Duez, 2005). At slow speaking rate speech duration was shown to move towards a more normal value (Hammen and Yorkston, 1996), making speech more intelligible (Ramig and Gould, 1986).

Comparable investigations have focused on the effects of articulation rate on the duration of segments and syllables in PD and normal speech. Comparisons of segment durations and vowel space across rate conditions in American English showed that the relative change in segment duration was similar, although PD patients tended to speak at faster rates (McRae, Tjaden and Schoonings, 2002). Rate did not strongly affect measures of acoustic space for vowels /i, æ, u and ɒ/ and fricatives /s and ʃ/ in PD speech as a whole although within each rate condition there was a tendency for vowel space areas for PD speech to be smaller than the space area for control speech. Similarly, in the so-called oral diadochokinesis tasks (i.e. reiteration of a given syllable as fast as possible on a single breath), the performance of most patients suffering from PD was similar to that observed in control subjects (Ackerman, Hertrich and Hehr, 1995; Ludlow, Connor and Bassich, 1987). This was shown to have been caused by slow speech gestures concomitant to an undershooting of articulatory gestures (Ackermann, Konczak and Hertrich, 1997).

Articulatory undershoot is a main characteristics of PD speech. It can be reflected at the acoustic level by consonant imprecision such as change of plosives into their fricative counterparts, omission of speech segments (Ackermann et al., 1993) and changes in syllable structure. For example, the omission of coda consonants in heterosyllabic [C1#C2]'s or C2 or C1 in homosyllabic [C1C2]'s leads to syllable simplification with an increasing number of CV-syllables and a decreasing number of CVC's and CCV's (Duez and Viallet, 2003). Articulatory undershoot does not act uniformly and appears to be influenced by linguistic demands as in normal speech. For example, in an acoustic study of PD speech, Ackermann and Ziegler (1991) observed that the stop consonant closures associated with stressed syllables were performed at the expense of unstressed ones, thus preserving information crucial to lexical access. Furthermore, PD patients were found to retain the normal pausing pattern, the link between distributional scheme of pauses and linguistic structure, thus helping listeners integrate the syntactic information contained in the message (Duez, 2005).

The above results suggest that PD affects temporal speech patterns in a complex way, compounded by speech duration being influenced by the phonological structure of languages. Consequently, investigating the impact of PD on the temporal organization of speech in different languages should not only permit a better understanding of how basal ganglia dysfunction impairs temporal speech processing but also clarify language-specific effects. In this context, the present investigation reviewed how PD affects the duration and the structure of syllables in French, a language whose syllable structure has a high proportion of open to closed syllables, a pattern of vowel reduction where most unaccented vowels are full vowels, and a pattern of accentuation with final prominence (Delattre, 1965-66; Di Cristo, 1984, Fletcher, 1991; Vaissière, 1991). Final lengthening effect is an inherent characteristics of motor programming which offers speakers an extra fraction of timing during which a following phrase can be planned (Cooper and Paccia-Cooper, 1980), and signals the boundaries of linguistic units on the basis of durational differences, at least in European Languages. French is a non-lexical stressed language whose rhythmic pattern relies mainly on the prominence given to final syllables of syntactic phrases (Delattre, 1965-66; Di Cristo, 1984; Fletcher, 1991; Vaissière, 1991). Contrary to English, French has no early stress marked by extra loudness in a

word, as a consequence, the realization of final lengthening may be of crucial importance for the production and perception of French rhythm.

The current study had three main objectives. The first was to examine the effect of articulatory undershoot on syllable duration in French and to check whether patterns of syllable duration are similar in PD and normal speech, as observed by Ackerman and his colleagues. The second was to control whether phrase-final lengthening, ubiquitous in normal speech and a fundamental characteristic of French rhythm, is still intact in PD speech and in strong correlation with the syntactic structure of the message. Finally, the third objective was to identify patterns of segment omissions and changes in syllable structure. To achieve this, syllable durations and structures were compared in a standard text read by twelve French subjects with mild to moderate PD and twelve French healthy control subjects. Syllable durations were analysed as a function of position within the syntactic phrase (i.e. final and non final) and presence of silent pauses (prepausal or non prepausal). Regarding syllable structure, recurring patterns in segment omission were identified, the vulnerability of speech segments was examined as a function of the nature of consonants and vowels, also position within the syllable, i.e. in syllable onset, coda and location within clusters. It was anticipated that this information might be useful in improving understanding of how dysfunction in basal ganglia affects temporal organisation of speech.

METHOD

Subjects

The data for this study were collected from 24 French native speakers composed of 12 individuals (7 males and 5 females) diagnosed with Parkinson disease and 12 age and gender matched control speakers.

The PD participants were between 7 and 19 years post-PD diagnosis ($M=10$), selected by the Department of Neurology at the Hospital of Aix en Provence. All met the following criteria: (1) They were diagnosed as having mild to moderate idiopathic PD, (2) they had no histories of neurological, respiratory, laryngeal, speech and voice diseases or disorders, apart from those associated with PD, (3) they were being treated with L-Dopa and had no surgical treatment, (4) they were experiencing motor fluctuations in response to their treatment and (5) they had adequate vision with corrective lenses and claimed not to suffer from hearing loss. Subject profile including age, year of PD diagnosis and month and year of recording can be seen in Table 1. To make the effects of PD more salient, antiparkinsonian medications were withheld overnight and the first recordings started after at least 10 hours without medication. Before recording, the motor disability of each patient was assessed using the Unified Parkinson's Disease Rating Scale (UPDRS), especially, dysarthria severity as defined by item 18: 0: normal; 1: slight loss of expression, diction and/or volume; 2: monotone, slurred but understandable, moderately impaired; 3: marked impairment, difficult to understand; 4: unintelligible.

The twelve control subjects were non-neurologically impaired and had adequate vision with lenses and did not report problems of hearing. Their characteristics are also listed in Table 1.

Insert Table 1 about here

Speech sample and recording equipment

The read speech sample was an excerpt of “La chèvre de Monsieur Seguin” (A. Daudet, 1869). Each subject was asked to read at his normal speech rate. The selected text was written

on paper and held before subjects by a research assistant. High-quality recordings were obtained in a sound-treated room of the Hospital of Aix en Provence. The acoustic signal was transduced using an AKG C410 head mounted microphone and recorded directly onto a PC hard disk at a sampling rate of 20 KHz.

ANALYSIS

Transcriptions.

The author transcribed readings orthographically. Omitted, added and repeated syllables and segments were reported in the transcription.

Temporal measurements.

Temporal acoustic measures were obtained using the Praat program (Boersma and Weenik, 2000). Measurements were made on combined wideband spectrograms and oscillograms displayed on a screen, and by listening to selected segments of the waveform. The overall passage was segmented into pauses and articulated sequences, then each articulated sequence was segmented into syllables. Durations were obtained using the segmentation criteria defined by Autesserre, Perennou and Rossi (1989). For example, for an articulated sequence and a syllable beginning and/or ending with a vowel or a sonorant, the limits were the first pulse and/or the last pulse of the vowel and/or the sonorant. When the initial and/or final segment was a fricative the limits were the appearance and/or the cessation of noise. In case of an initial or a final occlusive, there were two different possibilities: a) when the occlusive was voiced the sound sequence began with the voice bar and ended with the voice bar or with the release of a visible burst; b) when the occlusive was unvoiced so the occlusion could not be separated from a preceding or following pause; if there was a visible burst the articulated sequence began with burst and/or ended with burst release.

Syllable location.

Syllables were classified as a function of location within phrases. There were two main groups : (1) final syllables located at the edge of major and minor phrases (as defined by Blanche-Benveniste et al., 1990) and (2) non-final syllables. Silent pauses are known to lengthen boundary syllables by about 25% (Klatt, 1975), therefore a distinction was also made between final syllables followed or not by a pause. In Southern French, there is a strong tendency to produce the so-called mute [ə] in word-final syllables; to isolate any such pause lengthening effect, syllables containing the vowel [ə] were treated separately. In the absence of a pause, syllables with [ə] were classified in the non-final group. Mean durations were calculated for each location in each group and for each speaker. The degree of lengthening was obtained for each group and each speaker by dividing the difference between the mean duration of non-final syllables and the mean duration of prepausal and non-prepausal final syllables by the duration of non-final syllables.

Syllable structure

Syllabification is highly dependent on context, speech styles and speakers. Thus, the analysis of syllable structure was based on surface syllables, i.e. syllables with their segmental information. In French, a same word can have different syllable structures depending on the prosodic and consonantal context and the realisation of the optional mute [ə]. For example, the

final syllable of the word “bonheur” (happiness) can have a CVC structure [nœr#] if there is a following pause or a word with a consonant at the onset, and a CV structure [nœ.ra.vek] if the following word begins with a vowel such as “avec” (with). Eight syllable structures were defined as follows, with V as a vowel and C as a consonant : V, VC, CV, CVC, CVCC, CCV, CCVC, CCVCC. To identify omission and addition of speech segments surface syllables were compared with abstract syllables. A segment was considered as omitted when there was no perceptible and no acoustic trace.

RESULTS

Insert Table 2 about here

Since final syllables of major phrases had an almost identical duration to those of final syllables of minor phrases, and control speech had only two minor phrases with a prepausal final syllable, a single group of final syllables was constituted.

As seen in Table 2, mean durations, standard deviations and coefficients of variation are very close in PD speech and control speech. This is particularly obvious for non-final syllables, which correspond to standard times for French (Duez, 1987). Final syllables are significantly longer than non-final syllables in both PD speech and control speech, and the degree of lengthening is also comparable. Non-prepausal final syllables are 50% and 43% longer than non-final syllables in PD speech and control speech respectively. Prepausal pre-boundary lengthening is slightly greater in PD speech (85%) than in control speech (82%); the difference with non-prepausal pre-boundary lengthening is 29% for PD speech and 18% for control speech, which is close to that already reported (Klatt, 1975). Prepausal syllables with [ə] in both groups exhibit lengthening whose magnitude is similar to that found for non-prepausal phrase-final syllables. A two-way ANOVA (2 groups X 4 positions) on syllable duration yielded a significant main effect of syllable position [$F(3, 5956)=991, p=0.0001$], no effect of disease [$F(1, 5956)=0.01, p=0.9$] and a significant interaction of both factors [$F(3, 5956)=2.8, p=0.03$].

Both groups show a high variability across speakers. In PD speech the range of mean duration of non-final syllables varies from 136 ms to 186 ms, for non-prepausal final syllables from 198 ms to 256 ms and for prepausal final syllables from 243 ms to 330 ms. For control speech mean durations have a similar range: from 140 ms to 178 ms for non-final syllables, from 199 ms to 283 ms for non-prepausal final syllables, and from 251 ms to 342 ms for prepausal-final syllables. Each speaker had a very low number of prepausal syllables with [ə], for example one PD patient had only one and one control speaker none. Therefore prepausal syllables with [ə] were not included in two-way ANOVA's performed to determine the effects of speakers and syllable position (12 speakers X 3 positions) on mean duration in PD speech and control speech. PD patients [$F(11, 2845)=5.1, p=0.0001$] and syllable position [$F(2,2845)=707, p=0.0001$] have significant main effects on syllable duration and the interaction of both factors is significant [$F(22, 2845)=2.7, p=0.0001$]. Similarly, control speakers [$F(11, 2888)=11, p=0.0001$] and syllable position [$F(2,2888)=758, p=0.0001$] have significant main effects on syllable duration, the interaction of the two factors is also significant [$F(22, 2888)=2.4, p=0.0002$].

All speakers had final lengthening. For PD speech, the mean degree for prepausal syllables is 84.5% (SD: 20), with a range from 122% to 58% whereas for non-prepausal syllables the mean lengthening is 44.7% (SD=10) with a maximum of 60% and minimum of 28%. In control speech, non-prepausal syllables are lengthened by 49% (SD: 11) and prepausal syllables by 84% (SD: 14) with a range of 69% to 30 % and 100% to 64 %, respectively.

Syllable duration as a function of syllable structure

Insert Figure 1 about here

As seen in Figure 1, syllable duration increases significantly with the number of consonants for both PD speech and control speech, although the average lengthening is about 50 ms less for PD speech. CVCC's are longer than CVC's, which in turn are longer than CV's whereas CCVCC's and VC's are not lengthened compared to CCVC's and V's respectively. There are two possible explanations: 1) consonants are shorter in PD speech and 2) there is a greater consonant omission in PD speech. This assumption remains to be tested in an investigation of consonant duration. A two-way ANOVA (2 groups X 8 structures) on syllable duration revealed a significant main effect of structure [$F(7,5948)=294$, $p=0.0001$]), no effect of group [$F(1, 5948)=0.15$] and a significant interaction of the two factors [$F(7,5948)=2.4$, $p=0.01$].

Omission of segments as a function of syllable structure

Insert tables 3a and 3b about here

As seen in Tables 3a and 3b, syllables with onset and/or coda clusters tend to be reduced both in PD and control speech, the proportion of omissions being higher in PD speech. For example, 53 out of 338 CCV's changed into CV's in PD speech, 18 out of 341 in control speech. Out of the 53 CCVC's, 4 were produced as CV's and 5 as CVC's by PD patients and 4 as CVC's by control speakers. The two CCVCC's were produced as CCVC (PDS: 1; CS: 2) and CCV (PDS:1) respectively. Regarding the 17 CVCC's in PD speech, 2 were simplified into CV's and 3 into CVC's; in control speech 3 out of the 15 CVCC's were produced as CVC's. Coda singletons were omitted in both groups. For example, 51 out of 316 CVC's were produced as CV's in PD speech and 13 out of 326 in control speech. Concerning VC's, 21 out of 48 were produced as V's in PD speech, 18 out of 51 in control speech. In contrast, the omission of onset consonants is totally PD-speech specific. PD patients produced 14 CV's and 8 CVC's as V's and as VC's and one CCV as V; they omitted both initial and final consonants in 4 CVC's. There were also three cases with vowel omission, which led to the merging of a CVCV sequence into CCV.

C1C2 sequences

Insert Table 4 about here

As seen in Table 4, PD speakers produced C1's more frequently than C2's in onset and coda C1C2's; in a few cases they omitted both C1 and C2. This repartition is not significantly different [$\chi^2=0.14$, $p=0.7$]. Omissions are more frequent in PD speech than in control speech; however, the omission pattern is similar in both groups [$\chi^2=0.08$, $p=0.77$].

Insert Table 5 about here

As seen in Table 5, fricative-glides clusters had the highest number of omitted C2's (26.7%), followed by occlusives-glides clusters. In control speech, fricatives-glides clusters also had the

highest number of C2 omissions (8.2%). The repartition is significant in PD speech [$\chi^2=14$, $p=0.01$], but not in control speech [$\chi^2=6$, $p=0.5$]. Out of the ten omitted C1's in PD speech, 9 were voiced fricatives [v] often followed by the glide /w/, the omitted cluster was [lɥ] in “lui” (him). In control speech the omitted C1 was [v] in “avoir” (to have).

Insert Table 6 about here

As seen in Table 6, occlusive-sonorant clusters (3 out of 5) tend to be more sensitive than fricative-sonorant clusters (4 out of 11) to omission processes in PD speech; in control speech, there is no clear tendency; however, the limited number of cases in both groups does not allow the drawing of conclusions.

Coda and onset consonants

Insert Table 7 about here

As seen in Table 7, no clear tendency emerges from the repartition of omitted initial consonants as a function of nature in PD speech [$\chi^2=0.1$, $p=0.1$]. Omitted sonorants were /l, r or n/, fricatives were voiced (mostly /v/). Concerning coda consonants in PD speech, the percentage of omissions is greater for fricatives (31%) and occlusives (22.2 %) than for sonorants (16.8 %). In control speech, the percentages are lower (Fricatives: 16.3%, Occlusives: 4.9% and Sonorants: 6.3%). Again, the limited number of cases does not allow us to draw conclusions.

GENERAL DISCUSSION

The principal finding of the present study is the high degree of similarity between PD patients and control speakers in the pattern of syllable duration. This may seem paradoxical to the extent that PD is characterized by bradykinesia, i. e. slowness of articulatory movements, which should normally lead to longer syllable durations. However, it is in complete agreement with previous results on repetition syllable rates (Ackermann and Ziegler, 1991; Ackermann, Hertrich and Herh, 1995). In those studies, acoustic and kinematic data provided evidence that PD patients compensate for orofacial bradykinesia by reducing the amplitude of articulatory movements. The current study reinforces the supposition that the similar duration of syllables in PD speech results from a combination of articulatory undershoot and slowness of speech gestures.

In extreme cases, articulatory undershoot causes the complete omission of speech segments. In the present study, 6.2% of the produced syllables had an omitted segment in PD speech whereas the equivalent rate in control speech was only 1.6%. Omitted segments were mostly coda consonants and/or the second member of C1C2 sequences. The reduction of coda consonants is a well-known weakening process which results from a natural basic tendency to produce segments with less articulatory force and precision at the end of words and syllables (Straka, 1964). The decrease of articulatory strength concomitant with the reduced amplitude of movements which characterises PD speech can be expected to accentuate the omission of final consonants. Concerning C1C2's, the dominance of C1's is a well known tendency (Duez, 1998; Barry and Andreeva, 2001). This may be due to the fact that C1's are produced with more effort than C2's. The higher number of deleted C2's observed in PD speech than in control speech suggests either a strong reduction in the amplitude of speech movements or a difficulty switching between individual movements within sequences.

Omitted onset consonants were observed in PD speech: reduced mobility of the articulators may give rise to an inability to produce required speech movements. Similar omissions were reported in a kinematic analysis of orofacial movements by means of electromagnetic articulography (Ackermann et al., 1993). In that study, a subgroup of PD speakers showed difficulties, i.e. freezing during repetitive movements; this was characterised by the production of a sustained /a/ instead of the required repetitive consonant-vowel sequence /ta/. As /pa/ sequences were not affected, it was suggested that the differential impairment of the syllables /pa/ and /ta/ reflected the different mechanical properties of lower lips and tongue respectively. In the present study, 25% of the omitted initial consonants and the majority of C1's in C1C2's were voiced fricatives (/v/) often in /vw/ or /vu/ sequences. This suggests a certain difficulty or incapacity when producing and/or maintaining the labial gesture, something that needs to be confirmed in a larger corpus.

Speech-segment omissions may result from disruptions of the motor commands used to specify individual movements and guide their fluent execution (Kent et al., 2000). This may reduce the capacity of producing complex gestures or coordinating strings of complex gestures such as labio-dental segments and consonant clusters. Alternatively, disruptions may occur at the motor programming level, i.e. processes that occur prior to speech motor execution (Spencer and Rogers, 2005) causing PD patients to have greater difficulty constructing a motor program for certain responses within sequences. The present acoustic data do not permit to identify which level might be compromised. Articulatory undershoot does not act equally on all speech segments, preserving those which carry information especially crucial for successful lexical access (Browman and Goldstein, 1990; Lindblom, 1990). The acoustic data gathered in the present investigation confirm that PD patients exhibit relatively normal patterns of reduction and omission, indicating that the linguistic integrity of underlying phonological structures is not compromised in PD.

Another relevant result is the normal production of final lengthening by PD speakers. Analyses of the kinematic pattern of final lengthening showed that final lengthening is a local tempo slowing down not accompanied by any significant difference in articulator displacement (Edwards, Beckmann, and Fletcher, 1990). The fact that final lengthening does not require stronger movements or increased effort and amplitude of articulators would explain why PD patients have no difficulty with final lengthening.

The marking of final lengthening at the edge of syntactic phrases is in complete agreement with previous findings on pause pattern (Duez, 2005). In French, final syllables are important landmarks because they impose a cadence on the listener for integrating information (Vaissière, 1991). Hence, the normal production of final lengthening is of crucial importance for the perception of prominence pattern of utterances. However, the strong correlation between final lengthening and syntactic structure seems to disagree with results on syntax comprehension in PD speech. For example, in tests of syntax, PD patients were found to be impaired on decoding the grammatical structure of sentences (Lieberman, Friedman and Feldman, 1990). The difficulty in sentence processing was greater for sentences with increased complexity (Grossman, 1999).

Final lengthening is a biological constraint which seems to characterise human activities (Fraisie, 1974); the knowledge of its phonological use is acquired very early (Konopczinski, 1986). Thus the present data, by showing it is preserved in PD patients, would suggest that basal ganglia are not involved in timing patterns of syntax processing. However, since it is well known that speech disturbances increase as PD progresses toward its later stages (Ramig and Gould, 1986), the present results, limited to patients with mild to moderate impairment, should be reinforced by a similar assessment of PD patients suffering from more severe dysarthria.

ACKNOWLEDGEMENTS

The samples used are taken from data initially created by Dr. F. Viallet and B. Teston, LPL., using funds allotted by a PHRC.

REFERENCES

Ackerman, H., Ziegler, W. : Articulatory deficits in parkinsonian dysarthria : an acoustic analysis. *Journal of neurology, Neurosurgery and Psychiatry* 1991; 54: 1093-1098.

Ackermann, H., Gröne, B.F., Hoch, G and Schönle, P.W. (1993) Speech freezing in Parkinson's disease: A kinematic analysis of orofacial movements by means of electromagnetic articulography, *Folia Phoniatrica* 45, 84-89.

Ackerman, H., hertrich, I and Herh, T. (1995) oral diadochokinesis in neurological dysarthrias, *folia phoniatrica and logopaedica*, 47, 5-23.

Ackerman, H., Konczak, J. and Hertrich, I. (1997) The temporal control of repetitive articulatory movements in Parkinson's disease, *Brain and language*, 56, 312-319.

Autesserre, D. ; Pérennou, G. and Rossi, M. (1989). Methodology for the Transcription and Labeling of a Speech Corpus. *Journal of the International Phonetic Association*, 19(1), 2-15.

Barry, W. J. and Andreeva, B. (2001) Cross-language similarities and differences in spontaneous speech patterns, *Journal of the International Phonetic Association*, 31(1), 51-66.

Blanche-Benveniste, C. Bilger, M.; Rouget, C. & Eynde, K. van den : Le français parlé. Etudes grammaticales. Sciences du langage Ps: Ed. du CNRS, 1990.

Boersma, P. and Weenik, D. (2000). *Praat, a System for Doing Phonetics by Computer, Version 3. 4 (Technical Report 132)* Institute of Phonetic Sciences of the University of Amsterdam, www.praat.org.

Browman, C.P. and Goldstein, L (1990). Tiers in articulatory phonology, with some implications for casual speech. In *Papers in Laboratory Phonology I: between the grammar and physics of speech*, (M. Beckman, editor), pp 341-376. Cambridge, G.B: The Cambridge University Press.

Canter, G. J. (1963). Speech Characteristics of Patients with Parkinson 's Disease: I. Intensity, Pitch and Duration. *Journal of Speech and Hearing Disorders*, 28(3), 221-229.

Cooper, W. E. and Paccia-Cooper, J. (1980). *Speech and Syntax*. Cambridge : Cambridge University Press.

Darley, F.L., Aronson, A.E. and Brown, J.R.(1969) : Differential diagnostic patterns of dysarthria. *Journal of Speech and Hearing Research*, 12: 249-269.

Daudet A. (1869). La chèvre de Monsieur Seguin. In *les Lettres de mon Moulin*.

Delattre, P. (1965) *Comparing the phonetic features of English, German, Spanish and French*, Julius Gross Verlag.

Delattre, P. (1966) Studies in French and comparative phonetics, *Selected papers in French and English*, Mouton, London, the Hague, Paris.

Di Cristo, A. (1984). *de la Microprosodie à l'intonosyntaxe*. Editions Jeanne Lafitte.

Duez, D. (1987) *Contribution à l'étude de la structuration temporelle de la parole en français*. Thèse de Doctorat d'état, Aix en Provence.

Duez, D. (1998) Consonant sequences in spontaneous French Speech, *Sound patterns of Spontaneous Speech*, ESCA Workshop, La Baume-les-Aix, pp 63-68.

Duez, D. (2005) Organisation temporelle de la parole et dysarthrie parkinsonienne, in Ozsancak, C. and Auzou, P. *Les troubles de la parole et de la déglutition dans la maladie de Parkinson*, pp. 195-213, Marseille : Solal.

Duez, D. and Viallet, F. (2003), The Effects of Time on Temporal Variables in Speech read by subjects with Parkinson disease: Preliminary results, *International Congress of Phonetic Sciences*, Barcelone, 1627-1630.

Edwards, J , Beckman, M.E. and Fletcher, J. (1990) The articulatory kinematics of final lengthening, *Journal of the Acoustical Society of America*, 89(1), 369-382.

Fletcher, J. (1991) Rhythm and Final lengthening in French, *Journal of Phonetics*, **19**, 193-212.

Fraisse, P. (1974) *Psychologie du rythme*, Paris : PUF.

Gräber, S., Hertrich, I., Daum, I., Spieker, S. and Ackerman, H (2002): Speech perception deficits in Parkinson's disease: underestimation of time intervals compromises identification of durational phonetic contrasts. *Brain and Language*, 82: 65-74.

Grossman, M. (1999) : Sentence processing in Parkinson's disease. *Brain and Language*, 40: 387-413.

Hammen, V. and Yorkston, K. (1996). Speech and Pause Characteristics Following Speech Rate Reduction in Hypokinetic Dysarthria. *Journal of Communication Disorders*, 29, 429-445.

Kent, R., Kent, J.F., Weismer, G. and Duffy, J.R. (2000) What dysarthrias can tell us about the neural control of speech, *Journal of Phonetics*, 28, 273-302.

Klatt, D. (1975) Vowel lengthening is syntactically determined in a connected discourse, *Journal of Phonetics*, 3, 129-140.

Konopczynski, G. (1986): *Du prélangage au langage : acquisition de la structuration prosodique*, Thèse de Doctorat d'Etat, Université de Strasbourg.

- Lieberman, P., Friedman, J and Feldman, L.S. : Syntax comprehension in Parkinson's disease. *Journal of Nervous and Mental Disease* 1990; 178: 360-366.
- Lindblom, B. (1990) Explaining phonetic variation: a sketch of the H and H theory. In *Speech production and speech modelling* (W. Hardcastle and A. Marchal, editors), **Vol 55**, pp. 403-439. NATO ASI Series. Dordrecht, Boston and London: Kluwer Academic Publishers.
- Ludlow, C.L., Connor, N.P. and Bassich, C.J. (1987) Speech timing in Parkinson's and Huntington's disease, *Brain and Language*, 32, 195-214.
- McRae, P.A., Tjaden, K., Schoonings, B. (2002) Acoustic and perceptual consequences of articulatory rate change in Parkinson disease, *Journal of Speech and Hearing Research*, 45, 35-50.
- Metter, E. J. and Hanson, W. R. (1986). Clinical and Acoustical Variability in Hypokinetic Dysarthria. *Journal of Communications disorders*, 19, 347-366.
- Ozsancak, C., Parais, A.M. and Auzou, P. (2002) Evaluation perceptive de la dysarthrie : presentation et validation d'une grille clinique, *Revue neurologique*, 158, 431-438.
- Ramig, L. O and Gould, W.J. (1986) Speech characteristics in Parkinson's disease, *Neurologic Consultant*, 4, 1-8.
- Seguier, N., Spira, A., Dordain, M., Lazar, P. and Chevrier-Muller, C. (1974) Etude des relations entre les troubles de la parole et les autres manifestations cliniques dans la maladie de Parkinson, *Folia Phoniatrica*, 26, 108-126.
- Solomon, N. P. and Hixon, T. J. (1993). Speech Breathing in Parkinson's Disease. *Journal of Speech and Hearing Research*, 36, 294-310.
- Spencer, K.A. and Rogers, M.A. (2005) Speech motor programming in hypokinetic and ataxic dysarthria, *Brain and Language*, to appear.
- Straka, G. (1964) L'évolution phonétique du latin au français sous l'effet de l'énergie et de la faiblesse articulatoire, *T.L.L., Centre de Philologie Romane, Strasbourg II*, 17-28.
- Vaissière, J. , (1991). Rhythm, accentuation and Final lengthening in French, in Sundberg, J. , Nord, L. and Carlson, R. *Music, Language and Brain*, pp 108-120, Houndsmills : Macmillan.
- Viallet, F. and Gentil, M. (2001) Les troubles de la production de la parole au cours de la maladie de Parkinson : la dysarthrie hypokinétique. In *Les dysarthries* (P. Auzou, C. Ozsancak, V. Brun, Editors). Pp. 153-160. Paris : Masson.
- Volkman, J. , Heft, H. , Lange, H. W. and Freund, H. J. (1992). Impairment of Temporal Organisation of Speech in Basal Ganglia Diseases. *Brain and Language*, 43, 386-399.

Patients	Age	Sex	Years post-PD	UPDRS In Off State	Dysarthria Severity	Control	Age	Sex
P1	57	M	12	34	2	C1	58	M
P2	52	F	11	58	1	C2	53	F
P3	43	M	12	30	1	C3	47	M
P4	60	M	8	44	1	C4	60	M
P5	67	M	19	61	3	C5	70	M
P6	51	F	12	10	1	C6	55	F
P7	52	M	7	40	1	C7	59	M
P8	72	F	8	42	2	C8	67	F
P9	57	F	7	52	3	C9	62	F
P10	59	F	10	29	1	C10	61	F
P11	58	M	11	24	1	C11	60	M
P12	57	M	10	44	1	C12	62	F

Table 1. Subject characteristics including age and gender, years post-diagnosis of Parkinson's disease (PD). The motor disability of each patient was assessed by means of Unified Parkinson's disease rating scale (UPDRS). Dysarthria severity was estimated with item 18 of the UPDRS : 0: normal; 1: slight loss of expression, diction and/or volume; 2: monotone, slurred but understandable, moderately impaired; 3: marked impairment, difficult to understand.

Table 2. Mean duration (M), standard deviation (SD) and number (N) of final syllables located at the edge of major phrases and non-final syllables, whether preceding a pause (+) or not (-) and syllables with a produced [ə] before a pause. Coefficients of variation (C of V) are reported for each group of syllables.

	PD Speech				Control Speech			
	M	SD	N	C of V	M	SD	N	C of V
Major phrase -	224.4	76	408	0.35	237.4	81	422	0.36
Major phrase +	287.9	74	333	0.25	292.3	86	319	0.29
Non Final	156.1	59.5	2142	0.37	158.6	57.8	2184	0.36
Final [ə] +	222.8	60.6	90	0.27	205	52.1	66	0.25
All	182.2	78.1	2973	0.42	185	79	2991	0.42

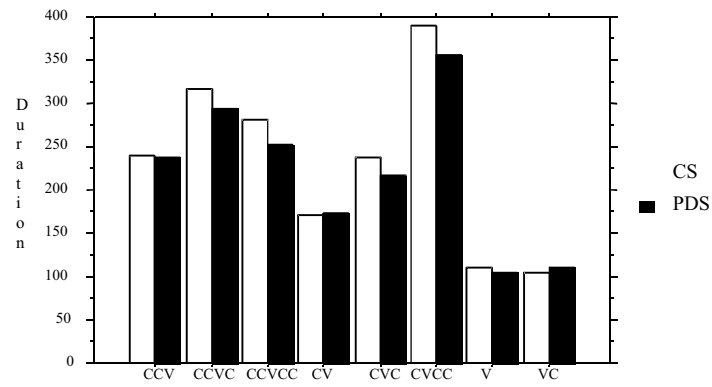


Figure 1. Mean syllable duration (in ms) as a function of structure in PD speech (PDS) and control speech (CS). Syllable structure is expressed as a function of consonant (C) and vowel (V).

Table 3 a and b. Matrice of omissions for PD speech and control speech as a function of syllable structure. Syllable structure is expressed as a function of consonant (C) and vowel (V). Produced syllables are compared with abstract syllables

3a	Abstract							
P		CCV	CCVC	CCVCC	CV	CVC	CVCC	VC
r	CCV	284	2	1	3	0	0	0
o	CCVC	0	42	1	0	0	0	0
d	CV	53	4	0	1960	51	2	2
u	CVC	0	5	0	3	257	3	3
c	CVCC	0	0	0	0	0	12	0
e	V	1	0	0	14	4	0	21
d	VC	0	0	0	0	4	0	22
	All	338	53	2	1980	316	17	48

3b	Abstract							
P		CCV	CCVC	CCVCC	CV	CVC	CVCC	VC
r	CCV	323	1	0	0	0	0	0
o	CCVC	0	49	2	0	0	0	0
d	CV	18	0	0	2001	13	0	0
u	CVC	0	4	0	0	313	3	0
c	CVCC	0	0	0	0	0	12	0
e	V	0	0	0	0	0	0	18
d	VC	0	0	0	0	0	0	30
	All	341	54	2	2001	326	15	51

Table 4. Number and % of C1's and/or C2's omitted in initial C1C2's and final C1C2's in PD and control speech (PDS and CS). Percentages are expressed as a function of the total number of syllables with initial and final clusters.

	PD Speech							Control Speech				
	C1		C2		C1+C2		Total	C1		C2		Total
	N	%	N	%	N	%		N	%	N	%	
Initial C1C2	10	2.5	52	13.3	1	0.2	392	1	0.7	16	4	398
Final C1C2	-	-	4	20	3	15	20	1	5.8	4	23.5	17

Table 5. Number of initial C1C2's with omitted (O) and non omitted (N O) C1's and/or C2's as a function of consonant nature (O: occlusive; F: fricative; S: sonorant; G: glide) in PD and control speech

	PD Speech				Control Speech		
	O			N O	O		N O
	C1	C2	C1+C2		C1	C2	
FS	1	4	-	31	-	2	39
FG	8	19	-	52	-	7	78
OS	1	11	-	104	-	3	111
OG	-	6	-	30	-	-	35
SG	-	12	1	112	1	4	116
FO	-	-	-	1	-	-	-
OO	-	-	-	-	-	-	1
SS	-	-	-	-	-	-	1

Table 6. Number of final C1C2's with omitted (O) and non omitted C1's and/or C2's as a function of consonant nature (O:occlusive, F: fricative and S: sonorant) in PD speech and control speech

	PD Speech			Control Speech			
	O		NO	O			NO
	C2	C1+C2		C1	C2	C1+C2	
FS	2	2	7	1	2	-	3
OS	1	2	2	-	-	2	5
SS	-	-	1	-	-	-	1
SO	-	-	-	-	-	-	1

Table 7. Number of omitted (O) and non omitted (NO) coda and onset consonants as a function of nature (O:occlusive, F: fricative and S: sonorant) in PD Speech and control speech

		Onset			Coda		
		O	F	S	O	F	S
PD Speech	O	3	3	9	19	12	50
	NO	826	608	801	42	32	247
Control Speech	O	-	-	-	3	9	20
	NO	-	-	-	58	46	295